REMARKS

Claims 1-52, 69-77, 80-82, 84-92, and 96-99 were pending in the present application. New claims 100-102 have been added, and claim 100 reads upon the elected invention as well as the elected species. Applicants note that new claims 101 and 102 depend from generic claim 99, which is believed to be allowable. Upon entry of the above amendments, claims 1-52, 69-77, 80-82, 84-92, and 96-102 will be pending.

Claims 1-52, 69-77, 82, and 84 have been withdrawn. Because claims 82 and 84 depend from generic claim 80 that is believed to be allowable, Applicants respectfully request that claims 82 and 84 to the non-elected species be rejoined, examined, and allowed. See 37 C.F.R. § 1.141.

Applicants thank the Examiner for the helpful telephonic interview that was conducted on December 12, 2007 during which the cited Skurkovich et al. (U.S. Patent No. 5,888,511) reference was discussed.

Reexamination of the application and reconsideration of the rejections are respectfully requested in view of the following remarks.

Claim rejections—35 U.S.C. § 102

Claims 80-81, 85-92, and 96-99 were rejected under 35 U.S.C. § 102 as being anticipated by Skurkovich et al. (U.S. Patent No. 5,888,511). This rejection is traversed for the reasons that follow.

Skurkovich et al. does not disclose effective treatment methods comprising the administration of a composition consisting of humanized or human monoclonal antibodies (or antigen-binding fragments thereof) against IFN-α alone (i.e., as the sole active ingredient) for the autoimmune diseases encompassed by claim 80, wherein no neutralizing anti-TNF antibodies are used in the method. Neither does Skurkovich et al. disclose effective treatment methods for the autoimmune diseases encompassed by claim 99 consisting of administration of a composition consisting of humanized or human monoclonal antibodies (or antigen-binding fragments thereof) against IFN-α alone (i.e., as the sole active ingredient) and the one or more of the other recited components. Although the Examiner has pointed to several portions of Skurkovich et al., when read in context these portions do not teach or suggest the method recited in claims 80 or 99.

For example, the Examiner states that Skurkovich et al. (at column 8, lines 32-33) "teach that [the] primary indicator of each autoimmune disease is the hyperproduction of IFN-α". (Office Action p. 4). However, the sentence immediately preceding the portion of Skurkovich et al. cited by the Examiner reads as follows:

> The present invention is based upon the inventors' conclusions that the optimal treatment of each different autoimmune disease or autoimmune condition involves the removal, neutralization or inhibition of complex pathological agents (including hyperproduced cytokines) from the patient, and/or the administration to the patient of an effective amount of selected molecules or antibodies, or their receptors, to bind to, neutralize or inhibit the circulating pathological agents and/or those on the surface of the cells targeted in the specific autoimmune response ("target cells").

Skurkovich et al., Column 8, lines 22-32 (emphasis added). Thus, while the portion of Skurkovich et al. cited by the Examiner states that the "primary indicator of each autoimmune disease is the hyperproduction of IFNα or, to be more exact, the disturbance of the synthesis of one or more alpha IFNs (IFNα comprises at least 15 distinct subtypes)", the immediately preceding sentence in Skurkovich et al. makes clear that "complex pathological agents" (rather than just the hyperproduced IFNα) must be removed, neutralized, or inhibited to treat autoimmune diseases. That is, this disclosure indicates that multiple agents must be used for effective treatment of autoimmune disease. This disclosure is consistent with the teachings of Skurkovich et al. at Column 4, lines 9-24 (cited in Applicants' previous submission), which states:

> ... because autoimmune diseases are complex, often characterized by multiple cytokine abnormalities, **effective treatment** appears to **require** the **simultaneous administration or utilization of several agents**, each targeting a specific cytokine pathway or its by-product. To meet this need, the methods of treatment of the present invention include not only the use of specific antibodies,. [sic] but also provide pleiotrophic autoimmune inhibitors, including antibodies to cytokines and HLA class II antigens, and antigens for the removal of autoantibodies to target cells or DNA. The use of these antibodies and antigens as disclosed in the present invention results in the removal, neutralization or inhibition of the pathogenic cytokine(s), HLA class II antigens, and/or autoantibody(ies) to target cells or DNA from the autoimmune patient, thereby significantly improving the quality of life of the individual.

Column 4, lines 9-24 (emphasis added).

The other portions of Skurkovich et al. cited by the Examiner do not modify these teachings in Skurkovich et al. that multiple agents are required for the effective treatment of autoimmune diseases. For example, the Examiner states that Skurkovich et al. "contemplate treating autoimmune disease in a patient comprising alone or in conjunction with administering to the patient an effective amount of one or more anti-IFN-α (column 6, lines 16+)." (Office Action, page 5). However, this paragraph of Skurkovich et al. describes a method of extracorporeal treatment for treating autoimmune disease "comprising removing autoantibodies from the patient by drawing fluid from the patient; passing said fluid over anti-TNF antibodies comprising an effective amount of different target cells, CD4 cells and/or DNA, to remove, neutralize or inhibit auto-antibodies in the patient's fluid; followed by returning the treated fluid to the patient." (Column 4, lines 16-29). The cited paragraph describes that the method of extracorporeal treatment may be conducted "alone or in conjunction with administering to the patient an effective amount of one or more antibodies". That is, this paragraph of Skurkovich et al. discloses that one or more antibodies may be administered **in addition to** utilization of extracorporeal treatment, which involves the use of anti-TNF antibodies comprising an effective amount of different target cells, CD4 cells and/or DNA. As discussed below, claims 80 and 99 exclude the use of anti-TNF antibodies, and these claims cannot be anticipated by the method of extracorporeal treatment in Skurkovich et al. cited by the Examiner.

Applicants submit that the only specific alleged treatments disclosed in Skurkovich et al. using antibodies against IFN-α as the sole active ingredient are in the context of rheumatoid arthritis and AIDS (see, e.g., *column 10, lines 9-13; Examples 3 and 7; column 3, line 57-column 4, line 9; and column 3, lines 15-39*).[1] In the extracorporeal treatment method described above, one or more antibodies may be administered **in addition to** utilization of the extracorporeal treatment, which comprises passing fluid from the patient over **anti-TNF antibodies** (see *Column 6, lines 16-35 of Skurkovich et al.*). These alleged treatments are not claimed in independent claim 80 (which excludes treatment of rheumatoid arthritis, AIDS, and diabetes as well as the use of anti-TNF antibodies) or independent claim 99 (which excludes treatment of rheumatoid arthritis, AIDS, and diabetes and recites a method "consisting of" administering the recited composition, thus excluding the extracorporeal treatment described in Skurkovich et al.).

[1] Applicants also note that Skurkovich et al. discloses alleged treatments using antibodies against agents/cytokines other than IFN-α (such as **anti-TNF-α**, **anti-interferon-γ**, and **anti-IgE**) as the sole active ingredient or agent in various contexts (see, e.g., column 4, lines 1-3; column 4, lines 34-37; column 4, lines 51-64; column 5, lines 18-31; column 6, lines 4-16; column 21, lines 33-36; column 22, lines 1-7; and Examples 3, 5, and 6).

To anticipate a claim, the reference must teach every element of the claim. *MPEP § 2133.*

Claim 80 is not anticipated because Skurkovich et al. does not teach treating an autoimmune disease that is not rheumatoid arthritis, AIDS, or diabetes using a method comprising administering a therapeutically effective amount of a composition **consisting of** one or more humanized or human monoclonal anti-IFN-α antibodies or antigen-binding fragments thereof and one or more of the other recited components, wherein no neutralizing anti-TNF antibodies are used in the method. In addition, claim 99 is not anticipated because Skurkovich et al. does not teach treating an autoimmune disease that is not rheumatoid arthritis, AIDS, or diabetes using a method **consisting of** administering a therapeutically effective amount of a composition **consisting of** one or more humanized, monoclonal anti-IFN-α antibodies or antigen-binding fragments thereof and one or more of the other recited components.

Therefore, because all of the limitations of claims 80 and 99 are not taught in Skurkovich et al., independent claims 80 and 99 (and claims 81-82, 84-92, 96-98, and 100-102 ultimately depending from claims 80 and 99) are not anticipated, and Applicants respectfully request that the rejection be withdrawn.

Information Disclosure Statement

Applicants have filed, concurrently with this Amendment, an Information Disclosure Statement in which the file history of Skurkovich et al. (U.S. Patent No. 5,888,511) has been cited. Applicants note that during prosecution of Skurkovich et al., the following pharmaceutical claim was submitted in the Preliminary Amendment dated December 22, 1997 (see page 5):

> 33. (Amended) A pharmaceutical composition [comprising an effective amount to treat] for treatment of a patient [with] having an autoimmune disease, said composition comprising an effective amount of one or more components selected from the group consisting of [: antibodies] an antibody to gamma interferon, [antibodies] an antibody to gamma interferon receptor, gamma interferon receptor, [antibodies] an antibody to alpha interferon, an antibody to to alpha interferon receptor, alpha interferon receptor, an autoimmune inhibitor, and a pharmaceutically acceptable carrier therefor.

Conclusion

For the foregoing reasons, claims 80-82, 84-92, and 96-102 are considered allowable. A Notice to this effect is respectfully requested. If any questions remain, the Examiner is invited to contact the undersigned at the number given below.

Respectfully submitted,

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